L6 ANSWER 5 OF 5 MEDLINE on STN DUPLICATE 2
AN 2001246638 MEDLINE
DN 21136237 PubMed ID: 11238588
TI Aberrant in vivo T helper type 2 cell response and impaired eosinophil recruitment in CC chemokine receptor 8 **knockout** mice.
AU Chensue S W; Lukacs N W; Yang T Y; Shang X; Frait K A; Kunkel S L; Kung T; Wiekowski M T; Hedrick J A; Cook D N; Zingoni A; Narula S K; Zlotnik A; Barrat F J; O'Garra A; Napolitano M; Lira S A
CS Department of Pathology, University of Michigan Medical School, Ann Arbor, MI 48105, USA.
NC AI43460 (NIAID)
NO1-AI55270 (NIAID)
SO JOURNAL OF EXPERIMENTAL MEDICINE, (2001 Mar 5) 193 (5) 573-84.
Journal code: 2985109R. ISSN: 0022-1007.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200105
ED Entered STN: 20010517
Last Updated on STN: 20010517
Entered Medline: 20010510
AB Chemokine receptors transduce signals important for the function and trafficking of leukocytes. Recently, it has been shown that CC chemokine receptor (CCR)8 is selectively expressed by Th2 subsets, but its functional relevance is unclear. To address the biological role of **CCR8**, we generated **CCR8** deficient (-/-) mice. Here we report defective T helper type 2 (Th2) **immune** responses in vivo in **CCR8**(-/)- mice in models of Schistosoma mansoni soluble egg antigen (SEA)-induced granuloma formation as well as ovalbumin (OVA)- and cockroach antigen (CRA)-induced allergic airway inflammation. In these mice, the response to SEA, OVA, and CRA showed impaired Th2 cytokine production that was associated with aberrant type 2 inflammation displaying a 50 to 80% reduction in eosinophils. In contrast, a prototypical Th1 **immune** response, elicited by Mycobacteria bovis purified protein derivative (PPD) was unaffected by **CCR8** deficiency. Mechanistic analyses indicated that Th2 cells developed normally and that the reduction in eosinophil recruitment was likely due to systemic reduction in interleukin 5. These results indicate an important role for **CCR8** in Th2 functional responses in vivo

6 ANSWER 2 OF 5 MEDLINE on STN
AN 2002734018 MEDLINE
DN 22384519 PubMed ID: 12496446
TI **CCR8** is not essential for the development of inflammation in a mouse model of allergic airway disease.
AU Chung Chan D; Kuo Frederick; Kumer Jeffrey; Motani Alykhan S; Lawrence Christopher E; Henderson William R Jr; Venkataraman Chandrasekar
CS Tularik, Inc., South San Francisco, CA 94080, USA.
SO JOURNAL OF IMMUNOLOGY, (2003 Jan 1) 170 (1) 581-7.
Journal code: 2985117R. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 200303
ED Entered STN: 20021227
Last Updated on STN: 20030306
Entered Medline: 20030305
AB Chemokine receptors play an important role in the trafficking of various **immune** cell types to sites of inflammation. Several chemokine receptors are differentially expressed in Th1 and Th2 effector populations. Th2 cells selectively express CCR3, CCR4, and **CCR8**, which could direct their trafficking to sites of allergic inflammation. Additionally, increased expression of the **CCR8** ligand, TCA-3, has been detected in affected lungs in a mouse model of asthma. In this study, **CCR8**-deficient mice were generated to address the biological role of **CCR8** in a model of allergic airway disease. Using two different protocols of allergen challenge, we demonstrate that absence of **CCR8** does not affect the development of pulmonary eosinophilia and Th2 cytokine responses. In addition, administration of anti-TCA-3-neutralizing Ab during allergen sensitization and rechallenge failed to inhibit airway allergic inflammation. These results suggest that **CCR8** does not play an essential role in the pathogenesis of inflammation in this mouse model of allergic airway disease.

L6 ANSWER 1 OF 5 MEDLINE on STN
AN 2003063921 MEDLINE
DN 22462119 PubMed ID: 12574386
TI Absence of **CCR8** does not impair the response to ovalbumin-induced allergic airway disease.
AU Goya Inigo; Villares Ricardo; Zaballos Angel; Gutierrez Julio; Kremer Leonor; Gonzalo Jose-Angel; Varona Rosa; Carramolino Laura; Serrano Alfredo; Pallares Pilar; Criado Luis Miguel; Kolbeck Roland; Torres Miguel; Coyle Anthony J; Gutierrez-Ramos Jose-Carlos; Martinez-A Carlos; Marquez Gabriel
CS Departamento de Inmunologia y Oncologia, Centro Nacional de Biotecnologia/Consejo Superior de Investigaciones Cientificas, Universidad Autonoma de Madrid, Cantoblanco, 28040-Madrid, Spain.
SO JOURNAL OF IMMUNOLOGY, (2003 Feb 15) 170 (4) 2138-46.
Journal code: 2985117R. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 200304
ED Entered STN: 20030208
Last Updated on STN: 20030426
Entered Medline: 20030425
AB Interaction of chemokines with their specific receptors results in tight control of leukocyte migration and positioning. **CCR8** is a chemokine receptor expressed mainly in CD4(+) single-positive thymocytes and Th2 cells. We generated **CCR8**-deficient mice (**CCR8** (-/-)) to study the in vivo role of this receptor, and describe in this study the **CCR8**(-/-) mouse response in OVA-induced allergic airway disease using several models, including an adoptive transfer model and receptor-blocking experiments. All **CCR8**(-/-) mice developed a pathological response similar to that of wild-type animals with respect to bronchoalveolar lavage cell composition, peripheral blood and bone marrow eosinophilia, lung infiltrates, and Th2 cytokine levels in lung and serum. The results contrast with a recent report using one of the OVA-induced asthma models studied here. Similar **immune** responses were also observed in **CCR8**(-/-) and wild-type animals in a different model of ragweed allergen-induced peritoneal eosinophilic inflammation, with an equivalent number of eosinophils and analogous increased levels of Th2 cytokines in peritoneum and peripheral blood. Our results show that allergic diseases course without critical **CCR8** participation, and suggest that further work is needed to unravel the in vivo role of **CCR8** in Th2-mediated pathologies.